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SUMMARY

1. Paroxysmal auricular fibrillation in patients with atherosclerotic cardiosclerosis develops most frequently in connection with acute transitory, or more prolonged, disturbances of the coronary circulation. Other factors, as pneumonia, disturbances of the gastrointestinal tract, etc., play a less frequent role.

2. In these cases, the anatomo-physiological basis for paroxysmal auricular fibrillation is either transient ischemia of the auricular muscle, overexertion of the auricular muscle in connection with the impairment of circulation in the ventricular muscle, or their overexertion due to a diminished output in the lesser circulation during inflammatory pulmonary processes. It is quite possible that there is a reflex action on the auricular muscle by different internal organs. Any one of these factors, or a combination of them, can bring about the development of multiple foci of excitability in the auricular muscle.

AUTHOR

Auricular fibrillation is one of the most frequent disorders of the cardiac rhythm in patients with atherosclerotic cardiosclerosis, mitral stenosis, and thyrotoxicosis. It occurs less frequently in other illnesses.

According to M. Ia. Ar'ev, A. F. Tur, S. I. Umanskii, and A. G. Plotitsina, A. M. Sigla, M. E. Mandel'shtam and G. S. Zenzinov, S. V. Shestakov, B. I. Gorokhovskii and other authors, auricular fibrillation can be recorded in 13.3-51.1 percent of patients with sclerotic heart disease. The paroxysmal form of auricular fibrillation, according to the same authors was found in another type of patient. Paroxysmal auricular fibrillation occurs more frequently in hyperthyroidism, and mitral stenosis. (M. Ia. Ar'ev, L. Ya. Siterman, S. V. Shestakov, and others).

This original communication presents a followup of 21 patients with atherosclerotic cardiosclerosis in whom paroxysmal auricular fibrillation was observed under clinical conditions; in three of these patients transient auricular flutter was diagnosed.

The purpose of this study is twofold: 1) to draw attention to the possibility of paroxysmal auricular fibrillation in patients with atherosclerotic cardiosclerosis; 2) to attempt to discover the underlying cause of these disorders.

There were 13 male and 8 female patients ranging from 41 to 69 years of age. All were diagnosed cases of coronary atherosclerosis and atherosclerotic cardiosclerosis. In 15 of those patients effects of cardiac sclerosis were noticeable (at rest and during exertion). Two of the patients developed myocardial infarcts, 1-3 months prior to admission to the hospital, and in one patient the infarct developed during inpatient status; 10 patients had concurring hypertension of the 2nd and 3rd stage. Four of the patients were admitted to the clinic with acute bronchopneumonia, and one with cancer of the right upper bronchus.

Complaints were usually within the limits of the basic accompanying disorder. Most (17) patients complained of pains in the cardiac region.

The case histories of eight patients revealed periodic, paroxysmal tachycardia occurring either shortly after the onset of pain in the heart region, after physical exertion, or after an intestinal disorder. The case histories of the rest of the patients revealed neither paroxysmal tachycardia nor other disturbances of the cardiac rhythm. The duration of the main complaints ranged from a few months to 14 years.

An objective examination in 17 patients revealed a cardiac enlargement in various degrees (1-2 cm, rarely more than 2), mainly to the left. In 18 patients dull heart sounds were detected, five had an apical systolic murmur, and in six patients an accentuated second sound over the aorta was heard.

In 8 patients electrocardiograms (ECG) taken soon after admission show signs of auricular fibrillation (tachysystolic form), and in one patient auricular flutter with a coefficient of incomplete transmission of impulses (2:1). The remainder of the patients had a sinus heart rhythm of a systolic frequency from 55 to 85 per minute. In five patients, during the first examination, instead of a sinus rhythm, various deviations of the P wave were observed: widening to 0.12-0.14 per second with decrease in amplitude and segmentation. In two of these patients the P-Q interval was simultaneously shortened (to 0.10-0.11 second) together with a normal duration and form of the QRS complex.

The above-mentioned ECG changes in the P waves of patients with clinical symptoms of coronary atherosclerosis indicate dystrophic, and possibly minor focal changes in the auricular muscle. It is to be expected that conditions impairing the coronary blood flow in the auricular muscle in such patients can further promote the appearance of multiple foci of excitability.

During the first examination of four patients with auricular fibrillation, low voltage QRS and T waves were concurrently established in standard and chest leads, which indicated diffuse changes in the auricular and ventricular muscles.

ECG tracings of coronary insufficiency were found in 12 patients. These were: moderate and sharp decrease in RS-T segment, flattening or inversion of the T wave in leads from the extremities and left chest. In one female patient major focal notched changes were detected in the zone of a transmural (septum atriorum and apical) myocardial infarct which occurred one month before admission to the clinic. Among eight patients suffering from hypertension and coronary atherosclerosis, only one developed hypertrophy of the left ventricle as determined by an ECG.

Clinical and ECG observations of patients during their stay in the clinic showed that in all cases the auricular fibrillation (tachysystolic form) was of a transient character. The duration of attacks varied from a few minutes to nine days. In 10 patients auricular fibrillation could be observed during a period of a few days followed by a reestablished steady sinus rhythm.

The detailed study of case histories, clinical findings and ECG are of great value in solving therapeutical problems, and in clarifying the pathogenesis of transitory auricular fibrillation in patients with atherosclerotic cardiosclerosis.

Development of dystrophic changes in the auricular muscle under various pathological conditions gives rise to an appearance of multiple foci of excitability, and consequently, to auricular fibrillation. However, dystrophic changes by themselves, sometimes markedly accentuated, are not the primary cause of arythmia.

The followup of patients with atherosclerotic cardiosclerosis indicates that different factors imposing a burden upon the auricular muscle, greatly in excess of normal, can precipitate a paroxysm of auricular fibrillation. We were able to establish in 12 patients the sequence of the appearance of auricular fibrillation shortly after an attack of stenocardia or during the period of development of a myocardial infarct. The onset of paroxysmal auricular fibrillation shortly after intensive and prolonged pains in the heart region is illustrated by the following observation:

Patient K., age 52, a streetcar conductor, was an inpatient from 2 October to 2 December, 1957, due to coronary atherosclerosis with attacks of stenocardia, atherosclerotic cardiosclerosis and paroxysmal auricular fibrillation. Since the spring 1957, the patient had constrictive pains in the precordial region. On the first and second days of October they were prolonged, at times very intense, with acute weakness.

The cardiac pains persisted during the first two days after admission. Observations on admission: a rhythmical pulse, 66 strokes per minute, blood pressure 135/80 mm. Other internal organs normal. General blood and urine analyses normal.

ECG on 10 October (Figure 1) shows a sinus rhythm, 63 systoles per minute and tracings of a moderate insufficiency of the coronary circulation in the area of the posterior-lateral wall of the left ventricle (negative T_3, flattened T_{aVF} in vertical electrical heart position, low T_2, obliquely displaced downward RS-T in leads CR_5 - CR_6).

Auricular fibrillation confirmed by ECG appeared on 4 October. In comparison with the tracings of 3 October features of coronary insufficiency in the area of the left coronary artery (lowered T deflection in chest leads) are expressed more distinctly.

Until 21 October, 1957, the sinus rhythm alternated with auricular fibrillation. From 21 October a steady sinus rhythm was reestablished. The condition of the patient improved. The precordial pains subsided. ECG from 21 October, 1 November, and 16 November show a normalization of segment RS-T and wave T, indicating an improvement in the coronary circulation.

This data demonstrate the development of paroxysmal auricular fibrillation in connection with the impairment of coronary circulation in the area of the left coronary artery. With the improvement in circulation the heart rhythm became steadily normal.

In accordance with the data of G. G. Lang; A. M. Sigal; S. V. Shestakov; and others, the anatomo-physiological basis for auricular fibrillation during disturbances of the coronary circulation is a spreading of local ischemia in the auricular muscle, precipitating the development of multiple pathological foci of excitability.

Recurrent transitory impairment of the circulation ultimately brings about the development of dystrophic and minor focal changes in the auricular muscle expressed on ECG by various alterations in the P wave. The presence of these dystrophic changes facilitates the development of paroxysmal auricular fibrillation during repeated disturbances of the coronary circulation, or during any other conditions affecting the auricular muscle (overexertion, etc.).

Transitory auricular flutter (24 hours) with a fluctuating coefficient of impulse transmission was observed in one patient, L., 60 years old, in the acute stage of infarct of the posterior lateral wall of the left ventricle, and in another patient, Ts., 51, during an acute transitory disturbance of the coronary circulation in the region of the posterior lateral wall of the left ventricle.

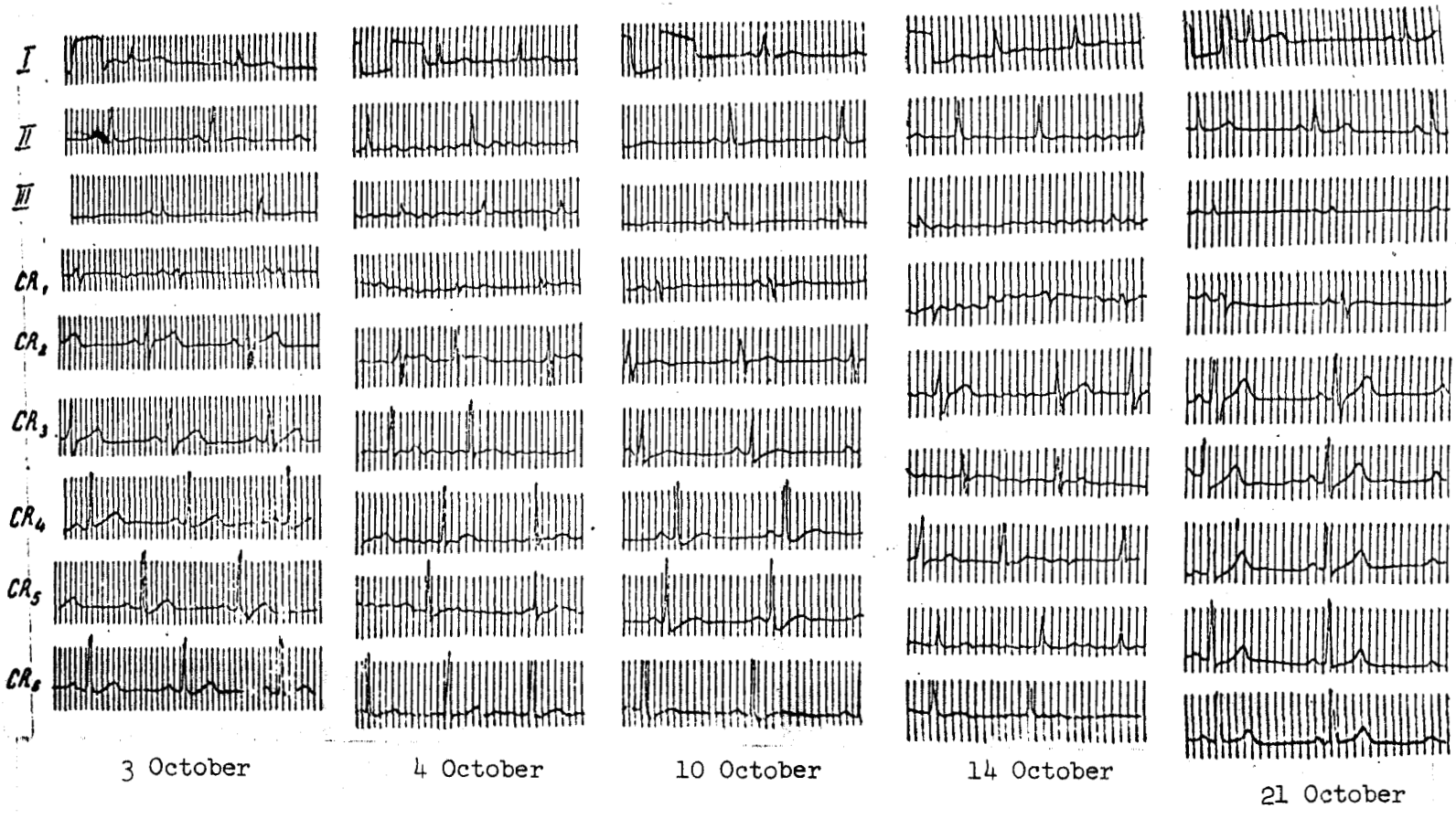

Figure 1. ECG of Patient "K"

The transitory auricular flutter in these cases can be explained by an increased hypoxia in the auricular muscle. With the restoration of circulation and limitation of the perifocal zone of necrosis, the sinus rhythm was reestablished in one of the patients with a myocardial infarct.

However, not only an impairment of the coronary circulation can lead to the development of a paroxysmal auricular fibrillation in atherosclerotic cardiosclerosis.

In four of our patients a transient auricular fibrillation was observed during a stage of acute pneumonia. These observations lead us to assume that pulmonary inflammation may have precipitated the development of auricular fibrillation; with the disappearance of the inflammatory processes in the lungs a correct sinus rhythm was reestablished in all of these patients.

Patient T., 65 years old, admitted to the clinic on 15 October, 1955, with residual symptoms of right lobar pneumonia, atherosclerotic cardiosclerosis, and obliterating endarteritis.

Observations on admission: temperature subfebrile, pulse regular--adequate fullness and tension, heart somewhat enlarged to the left and right, heart sounds dull. On percussion of the apices-tympanic sounds (hollow shading), over the right infrascapular area a soft reduction in percussion tone, respiration weakened, bubbly moist rales.

ECG dated 18 October (Figure 2) shows: sinus rhythm 55 per minute; the low voltage of the P wave in standard and chest leads rather unusual. The P wave borders directly on the beginning of the ascending bend of R deflection; interval P-Q equals 0.12 seconds, complex QRS equals 0.09 seconds; voltage of T wave normal in standard and chest leads. The disorders in the auricular complex of the ECG indicate changes in the auricular muscle.

October 22, seven days after admission, a sudden rise in temperature, to 39.7 C, cough reappeared, a white cell count of 11:300. Exacerbation of the lung condition. After 3 days auricular fibrillation appeared (tachysystolic form), persisting for 10 days. Parallel with the return to normal temperature and with the disappearance of the inflammatory changes in the lungs, a regular sinus rhythm was reestablished, and remained so during the entire period of the patient's hospitalization.

It is important to note, that with the cessation of the inflammatory process in the lungs the changes in the auricular complex, formerly observed, have also subsided, as is seen in Figure 2. Simultaneously with the reestablishment of a sinus rhythm, the voltage of the P wave increased, in standard and chest leads, and interval P-Q became normal. (0.15 sec).

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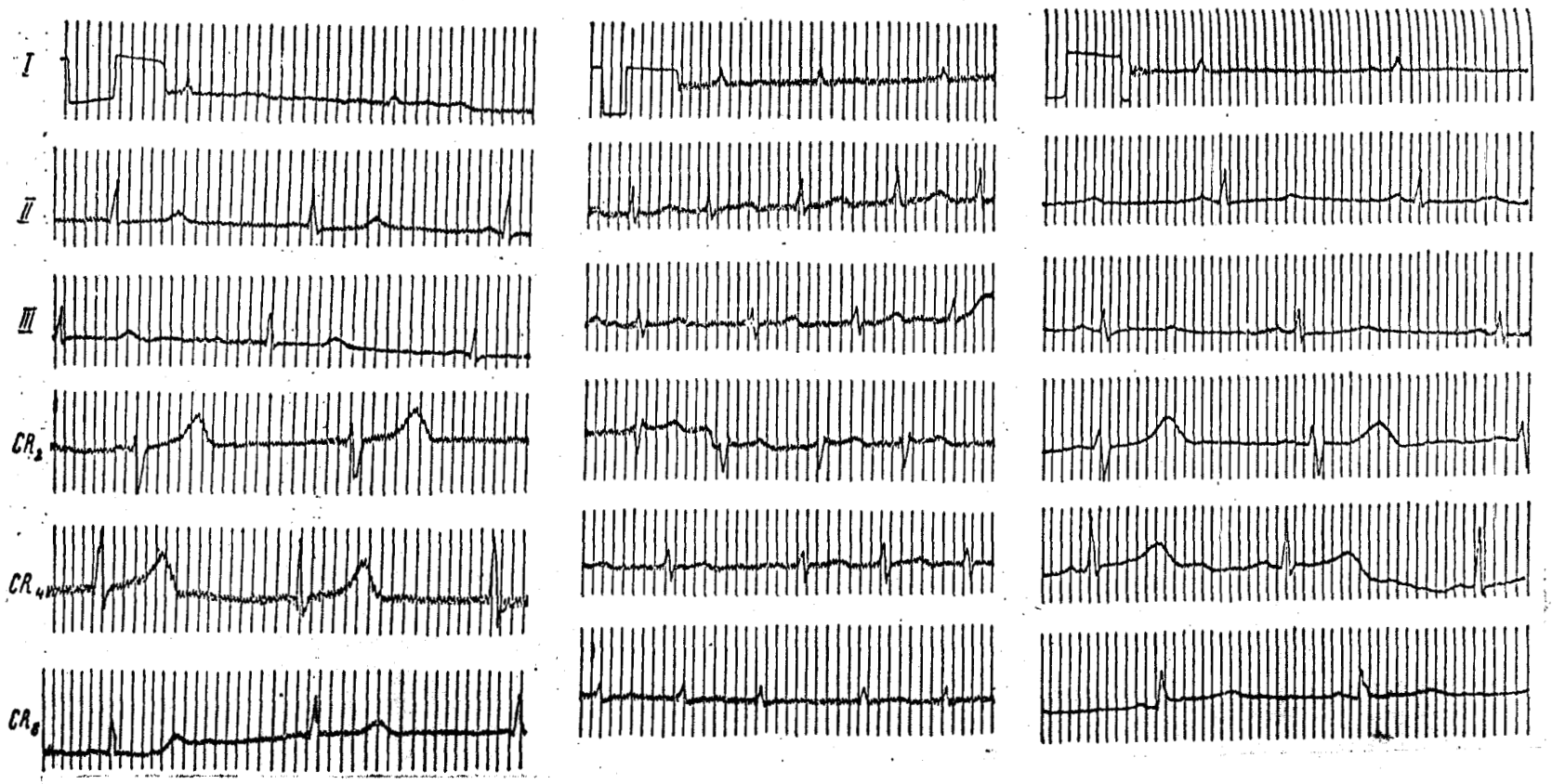

Figure 2. ECG of Patient "T"

Thus, these data vividly demonstrate the effect of pneumonia on the development of paroxysmal auricular fibrillation in an aged patient with atherosclerotic cardiosclerosis.

The development of paroxysmal auricular fibrillation during the period of acute inflammatory processes in the lungs (especially if they are of a disseminated nature), can be explained by an acute overexertion of the auricular muscle in connection with disturbances of the lesser circulation, and of blood arterialization. Evidently, reflex action from inflammatory zones of the lungs also has an effect on the auricular muscle.

The development of attacks of auricular fibrillation can also be explained by a reflex action of the vagus nerve shortly after an upset in the function of the digestive tract as seen in one of our 58-year old patients with coronary atheroclerosis.

In only three patients were we unable to find a relationship between the development of paroxysmal auricular fibrillation and other factors, of an extrinsic or intrinsic nature. These were patients more than 60 years old, with marked atherosclerosis, stenocardia (at rest and during exertion). Two of these cases had 3rd degree hypertension, and one was subject to a transmural myocardial infarct and thrombosis of the cerebral vessels with right hemiparesis, one month prior to admission. The development of paroxysmal auricular fibrillation in these patients is connected most likely with the chronic disturbance of coronary circulation, as verified by ECG in the clinic.

We were unable to establish any relationship at all between the recurrence of paroxysmal auricular fibrillation and hypertension. A similar form of auricular fibrillation was equally frequent in patients with atherosclerosis whether the hypertension was concurrent or not.

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